ACUTE PRIMARY CHOLECYSTITIS, WITH A REPORT OF FOUR CASES.

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Potain,[1] in 1882, and Courvoisier,[2] in 1890, described acute cholecystitis as a rare but rapidly fatal disease. In 1898 Richardson[3] published an article that should have convinced every practitioner that acute inflammation of the gall-bladder occurs not only as a complication of gallstones, or secondary to other diseases, but also as a primary and serious affection characterized by signs and symptoms that correspond to the extent and intensity of the pathological changes. I believe that most medical men are rather doubtful as to the occurrence of acute primary cholecystitis, and many still believe in such statements as the following: "Acute empyema, or gangrene of the gall-bladder, is associated with gallstones in 74 per cent. of the cases. In the remainder it occurs in zymotic diseases, as typhus fever, variola,"[4] etc. The time has come when we should discard such statements. We should know that acute primary cholecystitis does occur, that it can be diagnosticated, and that in most cases it can be treated successfully.

In this paper I shall discuss only cases of acute inflammation of the gall-bladder in which gallstones were not found, and in which the condition could not be considered secondary to any other disease.

Case I. *Sudden onset; chill; biliary colic; history of two primary attacks; operation; cholecystitis found; many adhesions; no gallstones; chronic appendicitis; recovery.*—Joseph B., a miner aged twenty-nine years, was seized with a violent chill. This occurred about midnight. Shortly afterward he began to suffer from violent paroxysmal pain in the abdomen. He vomited and the pain disappeared. In a few minutes the colic returned. The pain was very severe, was most intense in the pit of the stomach, and radiated to the right scapular region. Between midnight and 4 A.M. he had six paroxysms of pain. Examination (4 A.M., June 16, 1901) was as follows: The patient was in shock. Pulse rate, 110. There was no fever. His tongue was coated. The pupils were slightly dilated, but reacted normally. There was no jaundice. Examination of the heart and lungs was negative. The abdomen was slightly distended and was tender. The region of the gall-bladder was extremely tender. The right rectus was more tense than was the left. The remainder of the examination was negative. I washed out his stomach and gave him three-eighths of a grain of morphine and one-one-hundred-and-fiftieth of a grain of atropine hypodermically.

Previous History. The patient had measles when a child. He had never suffered from typhoid, variola, or pneumonia. Two years previously he was ill for one week with an attack similar to the present sickness. During this illness the pains were very severe, and he thought he had fever. Eight months ago he suffered from an illness that confined him to his bed for two weeks. The pain at this time was severe, and was paroxysmal for five days. Then it became continuous and gradually subsided at the end of two weeks. The region of the gall-bladder was tender for four weeks following this illness. The patient had never been jaundiced.

Operation. The morphine gave very little relief. The paroxysms of pain continued. At 8 A.M. his temperature was 101°, and his pulse rate 110. The patient was etherized at 9 A.M. After anæsthesia was complete I was unable to feel the gall-bladder or to determine that the liver was enlarged. An incision was made along the outer border of the right rectus muscle, extending from the costal arch downward. The omentum, colon, and gall-bladder were firmly matted together. I freed the gall-bladder and found that it was slightly enlarged, was very tense, and of a very dark color. I then brought the appendix into the wound. It showed evidences of chronic inflammation. After removing the appendix the gall-bladder was surrounded with gauze, aspirated, sutured to the transversalis fascia, and opened. It contained very dark and thick bile. The mucosa of the gall-bladder was œdematous and injected. No gallstones were found.

The patient made an uneventful recovery. The drainage was profuse, but contained no stones. The temperature and pulse rate became normal on the fourth day. The fistula was closed at the end of six weeks, and the patient has been well since that time.

A smear from the gall-bladder contents showed the presence of a short bacillus that stained well with methylene blue.

CASE II. *Chill; colic; high temperature and pulse rate; local tenderness over the gall-bladder; operation; gall-bladder enlarged, tense, and showed beginning gangrene in one place; cholecystostomy; no stones found; recovery.*—George T., aged twenty-four years, was seized suddenly with severe abdominal pain. The pain was paroxysmal, was located in the epigastrium, and did not radiate. He vomited twice. He had a severe chill, but cannot remember when this occurred. He is of the opinion that the chill preceded the pain. I saw him, with Dr. B. B. Slick, of Ridgway, Colorado, twelve hours after the onset of his trouble. At this time his temperature was 104° and his pulse rate 146. The pain had ceased. The region of the gall-bladder was sensitive on deep pressure. The right rectus muscle was rigid. The gall-bladder could not be palpated. Dr. Slick told me that the paroxysms of pain were very severe for ten hours and that one-quarter of a grain of morphine hypodermically had no effect on them. The patient's previous history showed that he had never been sick before.

Operation. Local anæsthesia was used. The gall-bladder was exposed through a vertical incision. It was enlarged and distended. The fundus showed an area of beginning gangrene. The gall-bladder was aspirated, sutured to the transversalis fascia, and opened. The organ was sufficiently enlarged so that it could be brought far enough into the wound to suture beyond the gangrenous area. The gall-bladder contained dark, thick material.

The patient made a rapid recovery.

No bacteriological examinations were made.

CASE III. *Chill; colic; fever; local tenderness; vomiting; operation; gall-bladder not perforated; general peritonitis; gangrenous cholecystitis; no gallstones; death.*—Mrs. S., aged thirty-six years, complained of violent abdominal pain about 11 P.M. At about the same time she had a violent chill. She vomited several times. The pain was paroxysmal, was located in the pit of the stomach, and did not radiate. She was given one-quarter of a grain of morphine hypodermically every hour. One and one-quarter grains were given before she was relieved. She then slept for six hours. When she awoke the pain returned, but was less severe. After the first thirty-six hours the paroxysms of pain ceased and she complained only of a dull, continuous pain. She ceased vomiting at the end of twenty-four hours, and did not begin to vomit again until the sixty-fourth hour. The bowels moved on the second day in response to five grains of calomel and an enema. The temperature ran from 101° to 103°; the pulse rate from 110 to 120. There was no jaundice. She had rheumatism fourteen years ago, and for eight years had suffered dyspnœa after exertion. For five years she had suffered more or less from the pain and hemorrhage caused by a uterine fibroid. She had had no acute infectious diseases.

I saw the patient seventy-two hours after the beginning of the illness. At this time the temperature was 104° and the pulse rate 130. The abdomen was distended, rigid, tympanitic, and tender. The region of the gall-bladder was more sensitive than the remainder of the abdomen. Examination of the lungs was negative. The heart was enlarged transversely. A systolic murmur was present over the apex and in the axilla. There was a leukocytosis of 22,000. The urine was scanty and contained much albumin. The liver dulness was slightly increased in size.

Operation. A longitudinal incision was made over the gall-bladder. The liver was enlarged. The gall-bladder was somewhat enlarged and distended. It was very dark in color, but was not gangrenous or perforated. There were no adhesions. General suppurative peritonitis was present. The appendix was normal. No tumor was felt in the region of the pancreas. The gall-bladder was aspirated and found to contain pus. A rubber tube and strips of gauze were placed through the posterior abdominal wall, on the right side, to drain the peritoneal cavity. Then the gall-bladder was anchored and opened. No stones were found. The patient died from general peritonitis forty-eight hours after the operation was performed.

Autopsy. General suppurative peritonitis; acute cholecystitis and cholangitis; fibrous myocarditis, with fatty degeneration; parenchymatous degeneration of the spleen, liver, and kidneys; a moderate degree of chronic interstitial nephritis. Atheromatous areas were present in the aorta and in the coronary arteries. There was a fibroid involving the fundus of the uterus.

CASE IV. *Acute primary cholecystitis; gangrene of the gall-bladder, with perforation; general peritonitis; death; no gallstones found at operation or autopsy.*—Carl H., a brakeman, aged twenty-seven years, who had had no previous sickness, was suddenly taken ill with a chill and with violent paroxysmal pains in the abdomen. The pain radiated, at times, to the right scapular region. He vomited continuously. His temperature averaged 103° and his pulse rate 120. A small bowel movement was obtained about thirty-six hours after the onset of the disease. On the second day of his illness the pain became continuous. There was no jaundice. There was marked tenderness over the region of the gall-bladder. The abdomen was distended, tender, and rigid.

Operation, fifty-five hours after the beginning of the illness, showed a gangrenous and perforated gall-bladder, with general suppurative peritonitis. The appendix and pancreas showed no marked changes. The patient died thirty hours after the operation. No gallstones were found at operation or autopsy.

CASE V.—M. Richardson[3] reports a case of acute cholecystitis occurring in a man, aged forty years, whose only previous illness had been an attack of "inflammation of the stomach," five years previously. The patient complained of acute pain in the abdomen. Twelve hours afterward his temperature was 101°. The abdomen was rigid and was diffusely tender. These symptoms were most marked on the right side. Operation revealed acute cholecystitis. A cholecystostomy was done and the patient recovered. The colon bacillus was found in the gall-bladder.

CASE VI. (Reported by Richardson.[3])—A man, aged twenty-eight years, who had never been sick, complained of paroxysmal pains in the groins. A hypodermic of morphine did not give him relief. The temperature was 102° and the pulse rate 108. A laparotomy revealed a general peritonitis. The gall-bladder was gangrenous, but was not perforated. No gallstones were found. The patient died on the fourth day following the operation.

CASE VII. (Reported by Richardson.[3])—A man, aged fifty-six years, who had previously had some pain in the region of the gall-bladder, was suddenly taken ill with abdominal pain, vomiting, fever. Temperature 103° and pulse rate 100. The area over the gall-bladder was markedly tender. In this case the patient had accurately located the point of greatest tenderness without advice or suggestions. Cholecystostomy revealed acute cholecystitis. The gall-bladder contained bacilli and cocci. No gallstones were found. The patient recovered.

Case VIII.—W. A. Lane[5] reports a case of acute primary cholecystitis occurring in a man, aged fifty-four years, who had always been in perfect health. The patient was suddenly seized with severe paroxysmal abdominal pain, prostration, and constipation. Attempts to produce evacuation of the bowels were tried for three days, but were unsuccessful. Laparotomy showed the duodenum and a portion of the colon covered with a layer of lymph. The gall-bladder was much distended, but was not gangrenous or perforated. A cholecystostomy was done. The gall-bladder contained mucopus but no stones. The patient made a perfect recovery.

Case IX.—E. M. Pond[6] reports a case of acute primary cholecystitis occurring in a healthy man aged sixty-five years. The onset was sudden, the pain paroxysmal, and the abdomen very rigid and tender. The area over the gall-bladder was extremely tender. Operation revealed a gangrenous and perforated gall-bladder; general peritonitis; no gallstones. The gangrenous portion of the gall-bladder was removed and drainage employed. The patient recovered.

Case X.—A. J. Ochsner[7] reports a case of cholecystitis occurring in a woman aged fifty-two years. She was sick one week previous to being operated upon. During this time she had four attacks of severe pain in the epigastrium. The gall-bladder was found greatly distended with thick, dark bile. No gallstones were found. A cholecystostomy was done and the patient recovered.

Case XI.—Curtis[8] reports a case of acute fatal peritonitis in which operation revealed advanced cholecystitis without perforation as the cause. The presence of gallstones was not mentioned.

Occurrence. Acute primary cholecystitis is not such a rare condition as is generally supposed. Musser[9] states that the primary form of acute cholecystitis is more common than is usually thought. M. Richardson states that acute cholecystitis, without known pre-existing gall-bladder disease, is more common than intestinal obstruction. The reports of large numbers of cases of operations on the biliary apparatus show plainly that cholecystitis —acute, chronic, with and without gallstones—is not an uncommon disease. But these reports are of little value in determining the relative frequency of occurrence of the acute primary form. The experience of any man who does a large amount of surgery and little else does not give a fair estimate of the relative frequency of occurrence of diseases like acute primary cholecystitis, for the following reasons: The large majority of the patients, coming to the surgeon of large experience, have first passed through the hands of the general practitioner. Many of these cases are chronic. But the rule is that, although the general practitioner may not have made a diagnosis, he is usually reasonably certain that the case is a surgical one before he refers it to the "surgical specialist." The chronic sufferers may find their own way to the surgeon of large experience, but in the great majority of cases it is the general practitioner who directs the plan of procedure in the acute serious diseases. For these reasons, the man who does very little outside of operative work is at a disadvantage in regard to diseases like acute primary cholecystitis. It is impossible, at the present time, to determine the frequency of occurrence of acute primary cholecystitis. I am of the opinion that acute and subacute primary cholecystitis is much more common than any of us would believe.

ETIOLOGY. I am unable to make any positive statements in regard to the etiology. It is probable that the colon bacillus is most frequently the cause of the inflammation. It is possible that the typhoid bacillus may remain latent in the gall-bladder for a number of years and then cause an acute attack of cholecystitis. Hunner[15] reports such a case. These patients usually give a history of a previous attack of typhoid fever. However, Cushing[11] reports one case of gallstones, with acute typhoid cholecystitis, in which there was no history of a previous attack of typhoid.

It has not been positively determined how the micro-organisms reach the gall-bladder. It is generally believed that they come from the intestinal tract. The usual idea has been that they reached the gall-bladder through the common and cystic ducts. Cushing[12] states that in cholecystitis following typhoid the organisms get into the gall-bladder by way of the portal vein. I have thought that the mechanism of infection might be as follows:

When the colon bacillus finds its way into the portal vein it seldom passes beyond the liver. Many times it is excreted in the bile. Now some of these organisms, in being excreted from the liver, might be carried by the bile into the gall-bladder. Some of the organisms might lodge between the mucous folds of the cystic duct and excite an inflammation. The swelling of the lining of the duct would soon close the outlet of the gall-bladder. This would lower the resistance of the gall-bladder mucosa (Charco and Gombault[13] and Netter[14]), and, according to Leubuscher, do away with the antiseptic properties of the bile in the gall-bladder. (Cushing says that the bile is not only not antiseptic, but is a good culture medium.) If we accept the foregoing as the process by which the inflammation starts, we can easily see how distention, gangrene, and perforation of the gall-bladder, and peritonitis, with or without perforation, might occur, according to the virulence of the infection and the resistance of the patient.

Pathology. I can add nothing to the pathology that has not been given in the report of the cases. It would seem that the presence of pus in the gall-bladder occurs only after the disease has become well advanced, that gangrene of the gall-bladder usually begins at the fundus, and that a virulent peritonitis can and does occur without perforation of the gall-bladder (Robson).

The changes in the liver are comparatively slight. This seems reasonable if we accept the mechanism of the infection as described in the foregoing. The organisms would be prevented from attacking the common and the hepatic ducts on account of the smoothness of their linings and the flow of bile, while the stagnation of the bile and the presence of large folds in the mucosa would predispose the cystic duct to infection.

The general changes in the body correspond to those that accompany any acute inflammatory process.

Symptomatology. The number of cases of acute primary cholecystitis is too small to outline a definite symptomatology. I shall discuss the symptoms in the order that I believe they occur, and, at the same time, shall mention the relation of the symptoms to the pathological changes.

Chill. The chill may be severe, moderate, or absent. It can be expected in the most severe cases.

Pain. The pain may accompany, precede, or follow the chill. It is of sudden onset, is very severe, is paroxysmal at the beginning, and is located in the epigastrium. It frequently radiates to the right scapular region, and in one of Richardson's cases was situated as low as the groins. The pain, in some cases, is identical with biliary colic. It is doubtless due to the gall-bladder trying to empty itself. When the musculature of the gall-bladder exhausts itself the pain ceases, only to return in a short time. When the tension in the gall-bladder becomes great and its musculature exhausted, the pain becomes continuous. When gangrene of the gall-bladder occurs the pain ceases. I shall not discuss the mechanism of acute colicky pain in the region of the gall-bladder. There are so many contradictory and radical statements on this point, made without proof, that it is impossible to draw conclusions.

Vomiting. Vomiting may be expected in most severe cases. It is an early symptom. It usually ceases after the first twenty hours, but returns if peritonitis develops. No cases of fecal vomiting have been recorded.

Fever and Elevation of Pulse Rate. These are present in all cases. They occur shortly after the patient has recovered from the chill and the shock of the primary paroxysm. The intensity of these symptoms depends on the severity of the infection. They still are present after the gangrene has abolished pain and obscured local tenderness.

Local Tenderness. This is an early and constant symptom. The entire abdomen may be sensitive, but the rigidity and tenderness are most marked on the right side, and the point over the gall-bladder is most sensitive to pressure, except when the gall-bladder becomes gangrenous. A gangrenous gall-bladder is insensible.

Jaundice is not a symptom of cholecystitis. It may occur as a complication or as part of a pre-existing disease.

Enlargement of the gall-bladder and the liver usually cannot be determined.

Constipation. Constipation is usually present, but in most cases will yield to treatment.

The changes in the blood and urine correspond to those found in ordinary acute infections.

DIAGNOSIS. Cholecystitis is recognized by the sudden onset, the characteristic pain, the fever, and the local point of tenderness. Gangrene of the gall-bladder is strongly suspected when the pain and tenderness cease, or are markedly diminished, with the general symptoms increasing in severity. Collapse might follow perforation. Further reports will be necessary to determine this point. It is impossible to recognize gallstones when they have given no previous symptoms.

PROGNOSIS. Acute primary cholecystitis is amenable to successful surgical treatment in all cases in which operative treatment has not been unduly delayed. The mortality depends entirely upon the promptness of recognition and treatment. I am of the opinion that most of the cases would die if not treated surgically. Doubtless many patients who have died from "inflammation of the bowels" had acute primary cholecystitis at the beginning of their illness.

The subacute and the mild cases of acute primary cholecystitis recover. I believe that primary cholecystitis may occur in all degrees of severity, from the mild attacks that Musser believes to be followed by gallstones to the most severe ones that cause death in twenty-four to forty-eight hours.

TREATMENT. I know of no medical treatment for cholecystitis. If the patient refuses surgical aid, the stomach should be washed out, rectal feeding resorted to, morphine given if necessary, and cathartics avoided.

I am of the opinion that the advice of Ochsner not to operate during an acute attack of cholecystitis is dangerous. I believe that all acute cases should be operated upon as soon as the diagnosis is made. Cholecystostomy meets the indication in the early stages and is not so severe on the patient as is cholecystectomy. At present I should advise removal of the gall-bladder only in cases with extensive gangrene, and drainage of the gall-bladder in all other acute cases. The presence of stones does not modify the treatment. I believe that in acute infected cases cholecystostomy has a smaller mortality than has cholecystectomy. If the foregoing advice is followed, a secondary operation may occasionally be required.

This is not especially objectionable. Secondary operations on the gall-bladder are usually safe and are not complicated with acute infection.

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VARIETIES OF SPLENIC ANÆMIA.

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THIS report of five cases, with pathological studies in three of of them, is presented as a contribution to the knowledge of the condition generally designated as splenic anæmia. I have elsewhere expressed doubt regarding the individuality of such a condition, and the conviction that many different sorts of diseases have been classed under this name. For convenience of clinical terminology the name may, however, be retained, and perhaps some order may be established before long.

CASE I. *Male, aged fifty-nine years, history of dysentery(?), duration over two years, probably six years or more; stunted growth, pigmented skin, clubbed fingers, osteoarthritis, enlarged spleen; chloroanæmia with leukopenia, hemorrhages, diarrhœa; early enlargement followed by diminution in the size of the liver; numerous intercurrent infections; death in stupor; autopsy.*—L. C., a cigarmaker, aged fifty-nine years, native of Belgium, was admitted April 18, 1900. The history recorded at that time is as follows: His father died of phthisis, his mother of jaundice, one brother of rheumatism. He had jaundice twenty-two years before and gonorrhœa sixteen years before. He denied syphilis and malaria, but had been in Mexico and Africa with the French army and may have had malaria. He gave an uncertain account of some sort of diarrhœal